Table 15. Human pharmacology of nicotine

Primary effects*	Withdrawal symptoms
Pleasure	Irritability, restlessness
Arousal, enhanced vigilance	Drowsiness
Improved task performance	Difficulty concentrating, impaired task performance
Relief of anxiety	Anxiety
Reduced hunger	Hunger
Body weight reduction	Weight gain
	Sleep disturbance
	Cravings or strong urges for nicotine
Electroencephalogram desynchronization	
Increased circulating levels of catecholamines, vasopressin, growth hormone, adrenocorticotropic hormone (ACTH), cortisol, prolactin, beta-endorphin	Decreased catecholamine excretion†
Increased metabolic rate	
Lipolysis, increased free fatty acids	
Heart rate acceleration	Heart rate slowing†
Cutaneous and coronary vasoconstriction	
Increased cardiac output	
Increased blood pressure	
Skeletal muscle relaxation	

*Some of these effects are related in part to relief of withdrawal symptoms.
†May represent a return to baseline rather than true withdrawal.
Source: Benowitz 1992a.

Genetic differences in the number of nicotinic receptors and pharmacologic responses to nicotine have been well demonstrated in animals (Marks et al. 1991). Genetic differences in pharmacologic responses to nicotine could underlie different susceptibilities to nicotine addiction, as appears to be the case for certain types of alcohol addiction (Hughes 1986; Cloninger 1987; Carmelli et al. 1992). Genetic susceptibility may vary by ancestry of origin (for example, sickle cell disease and African American ancestry). Genetic differences in nicotine responsiveness associated with ancestry of origin remain to be explored.

Tolerance, Withdrawal, and Addictive Tobacco Use

With prolonged or repeated exposure to nicotine, neurologic changes (neuroadaptation) occur. In animals, chronic nicotine exposure results in an increased number of nicotinic receptors in the brain (Marks et al. 1985). During the course of these changes, the smoker develops more brain nicotinic receptors and an increased tolerance to the various effects of nicotine. For example, previous studies have shown that at autopsy, the number of nicotinic receptors was greater in the brains of cigarette smokers than in those of nonsmokers (Benwell et al. 1988). Smokers develop substantial tolerance to the behavioral arousal and cardiovascular effects of nicotine in the course of a single day (Benowitz et al. 1989b). They can regain sensitivity to the effects of nicotine, at least in part, after overnight abstinence from smoking.

As a consequence of these neurologic changes, nicotine withdrawal symptoms appear when nicotine use is abruptly stopped (Table 16) (Hughes and Hatsukami 1992). Withdrawal symptoms include restlessness, irritability, anxiety, drowsiness, impatience, confusion, impaired concentration, and depression (Hughes et al. 1990). Some abstaining smokers gain weight, and others have impaired performance measures, such as reaction time. Many abstaining

Table 16. Incidence* of nicotine withdrawal symptoms, United States

Symptom	Clinic attendees (%)	Self-quitters (%)
Anxiety	87	49
Irritability	80	38
Difficulty concentrating	73	43
Restlessness	71	46
Hunger	67	53
Craving	62	37
Nocturnal awakenings	24	39
Depression	NA	31

*Percentage of subjects with postcessation ratings greater than precessation ratings 2 days after they quit smoking.
NA = data not available.
Sources: Hughes 1992; Hughes and Hatsukami 1992. Adapted from Hughes and Hatsukami 1992.

smokers have a strong craving to smoke a cigarette. Most of the withdrawal symptoms reach maximal intensity 24 to 48 hours after cessation and gradually diminish in intensity within three to four weeks (Gross and Stitzer 1989; Hughes et al. 1990), although some individuals experience longer lasting symptoms (USDHHS 1988). These symptoms, which also appear after quitting the use of smokeless tobacco (CDC 1994) or nicotine gum, are relieved following the administration of nicotine—a strong indication that the withdrawal symptoms are related to the effects of nicotine.

The degree of nicotine dependence is determined in part by the level of nicotine that accumulates in smokers. In general, the level of accumulated nicotine is proportional to the number of cigarettes smoked per day. Consistent with the concept of a daily tolerance-withdrawal cycle, a short duration of time between awakening and smoking the first cigarette is associated with a high degree of nicotine dependence (Heatherton et al. 1989). This presumably reflects an effort to relieve nicotine withdrawal symptoms. These two factors—the number of cigarettes smoked per day and the amount of time from awakening to smoking the first cigarette—are commonly used to assess the severity of nicotine dependence (Fagerström and Schneider 1989).

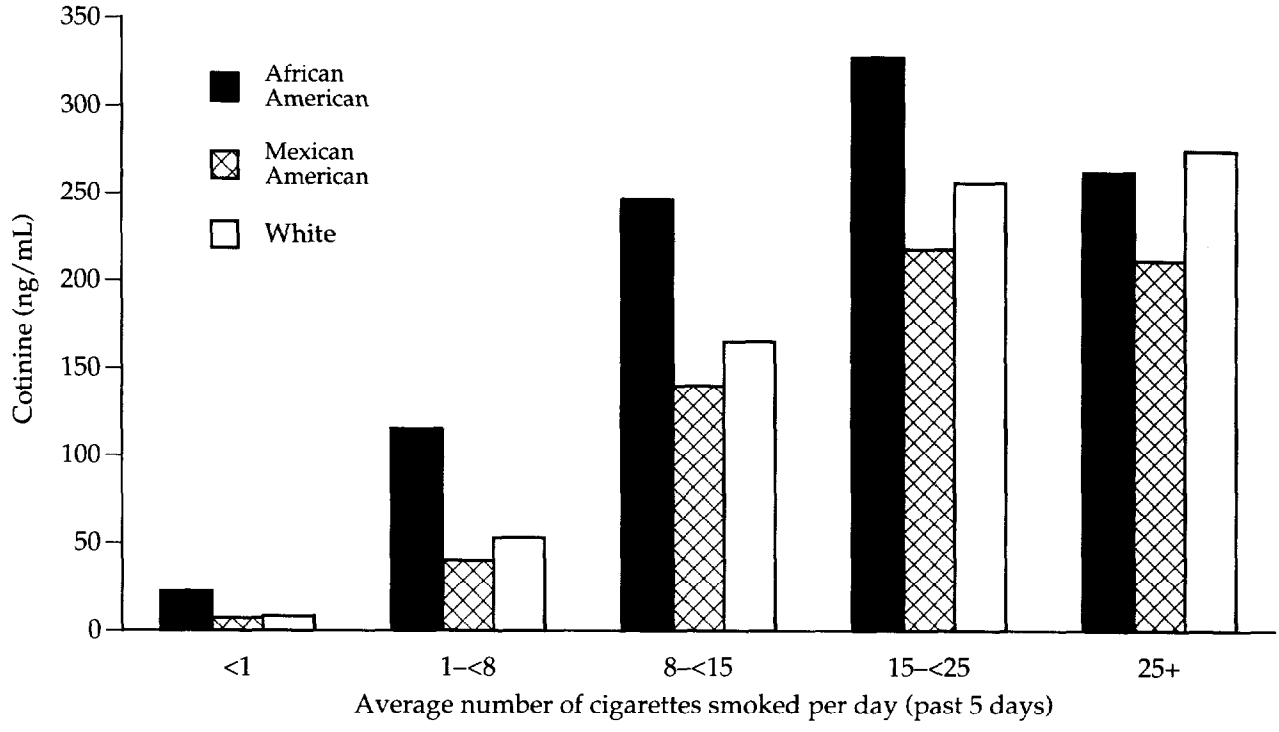
Level of Addiction

Assessments of the level of nicotine addiction help predict responses to nicotine and serve as a potential guideline for therapeutic approaches to smoking cessation. The professionals who design strategies to prevent tobacco use and treat persons with nicotine addiction need to understand the high level of addiction among cigarette smokers and to appreciate the group-specific cultural characteristics of the behavior and smokers' individual reasons for initiating, continuing, and quitting tobacco use (Krasnegor 1979; Grunberg and Acri 1991). The most widely used indexes of addiction levels are the number of cigarettes smoked per day, the serum nicotine or cotinine level, the Fagerström dependence questionnaire (Fagerström and Schneider 1989), and the diagnostic criteria of the *DSM-IV*™ (APA 1994). The Fagerström dependence questionnaire incorporates questions about the number of cigarettes smoked per day, the time between awakening and smoking the first cigarette of the day, as well as episodes in which the smoker lost control of smoking behavior (such as smoking at inappropriate times or in inappropriate places). The prevalence of smoking cessation—and conversely, the number of unsuccessful quit attempts—also reflects the level of addiction, at least in part. The brand of cigarette smoked might be expected to correlate with a person's level of dependence because high-yield cigarettes nominally deliver more nicotine per cigarette. However, in large surveys of smokers, only a modest relationship was found between yield (measured by a smoking machine) and levels of nicotine or cotinine in the body (Benowitz et al. 1986; Coultas et al. 1993). This is because people smoke differently than machines that are set to a standardized testing protocol—that is, they are able to take more frequent or deeper puffs, to smoke each cigarette more completely, to smoke more cigarettes per day, and to block ventilation holes in the cigarettes (Henningfield et al. 1994; NCI 1996a).

Racial/Ethnic Differences in Nicotine Metabolites

Evidence suggests that African Americans have higher cotinine levels per reported number of cigarettes smoked per day than whites (Wagenknecht et al. 1990; English et al. 1994; Clark et al. 1996a) (Figure 5). In Figure 5, the racial/ethnic minority group comparisons among those who smoked 25 or more cigarettes per day may be somewhat biased, because the average daily consumption for whites was substantially higher than that for African Americans and Mexican

Figure 5. Serum cotinine levels by number of cigarettes smoked daily for African Americans, Mexican Americans, and whites, National Health and Nutrition Examination Survey, United States, 1988–1991



Note: N = 2,136.
Source: National Center for Health Statistics, public use data tape, 1997.

Americans. Clark and colleagues (1996b) found no evidence that underreporting of daily cigarette consumption occurred more often in African American than in white smokers.

One possible explanation for the higher cotinine level among African Americans is that African Americans may absorb more nicotine from their cigarettes than whites (Benowitz et al. 1995). Greater absorption could result from several factors, including group-specific patterns of smoking behavior (i.e., more and deeper puffs per cigarette or longer retention of tobacco smoke in the lungs) (Benowitz et al. 1995). Additionally, menthol in cigarettes may facilitate absorption of cigarette smoke constituents (Jarvik et al. 1994; McCarthy et al. 1995; Clark et al. 1996a). However, the fact that African Americans smoke menthol cigarettes more commonly than whites do explains only a small percentage of their higher levels of cigarette smoke constituents (Wagenknecht et al. 1992; Ahijevych et al. 1996; Clark et al. 1996a).

Racial/ethnic differences in nicotine metabolism could influence the development of nicotine addiction. Several researchers have suggested that African Americans might metabolize cotinine differently than whites (Pattishall et al. 1985; Wagenknecht et al. 1990; English et al. 1994; Benowitz et al. 1995). Results of studies of nonsmokers support this hypothesis (Pattishall et al. 1985; Wagenknecht et al. 1993; Crawford et al. 1994; Knight et al. 1996; Pirkle et al. 1996). Most of these investigations (Pattishall et al. 1985; Crawford et al. 1994; Knight et al. 1996; Pirkle et al. 1996) reported that African Americans had higher cotinine levels than whites, even after ETS exposure and other factors were taken into account. These findings may be limited by the fact that no measures of tobacco smoke or nicotine concentrations in the air were obtained.

Based on a preliminary report of data for 40 African Americans and 39 white controls matched for age, gender, and cigarette consumption, Benowitz and colleagues (1995) reported that the disposition kinetics of nicotine were similar for both groups. For example, the percentage conversion of nicotine to cotinine was similar across groups. However, the clearance of cotinine was significantly lower for African Americans than for whites. Additionally, the average estimated intake of nicotine per cigarette smoked was 1.41 mg in African Americans and 1.09 mg in whites. This difference is of borderline statistical significance ($p = 0.07$) (Benowitz et al. 1995). African Americans took in 28 percent more nicotine per cigarette than would have been expected based on FTC yields; whites took in 9 percent more nicotine per cigarette than would have been expected based on FTC yields (Pérez-Stable et al., unpublished data).

Investigators have also found cotinine levels in African Americans that were higher than expected for the number of cigarettes smoked. Ahijevych and Wewers (1993) found an average salivary cotinine level of 402 ng/mL in African American women who smoked an average of 15 cigarettes per day. This level is much higher than the expected level found in other persons who smoked the same number of cigarettes. Clark and colleagues (1996b) reported that African American smokers smoked longer cigarettes and more of each cigarette than white smokers. However, because they smoked fewer cigarettes each day, African Americans smoked fewer total daily millimeters of cigarettes. Among young adults in the CARDIA study, African Americans (48 percent) were more likely than whites (36 percent) to report that a substantial amount of their cigarette burned without their smoking it (Wagenknecht et al. 1992). Also, in a study of 33 African American and white women, Ahijevych and colleagues (1996) did not find a racial/ethnic difference in total puff volume (per cigarette).

Pérez-Stable and colleagues (1990) reported that among Mexican Americans who were part of the 1982–1984 HHANES, cotinine levels were unexpectedly high in smokers reporting low levels of cigarette consumption. Higher-than-expected cotinine levels may reflect underreporting of smoking by Hispanics, but the possibility also exists that Hispanics absorb or metabolize nicotine differently than whites (Henningfield et al. 1990). However, recent data from NHANES III (Figure 5) indicate that, among persons who smoked at least one cigarette daily, Mexican American smokers had lower serum cotinine levels in each consumption category than African American and white smokers.

Racial/Ethnic Differences in Self-Reported Nicotine Dependence

The use of questionnaires to systematically investigate racial/ethnic differences in nicotine dependence has been limited. Data from the 1987 NHIS (Table 17) show that African Americans were more likely than whites and Hispanics to report smoking their first cigarette of the day within 10 minutes of awakening, although these differences tended to disappear among those who reported smoking 25 or more cigarettes per day (NCHS, public use data tapes, 1987). Telephone survey data on smoking, collected as part of the Community Intervention Trial (COMMIT) for Smoking Cessation, also indicate that African Americans were more likely than whites to smoke within 10 minutes of awakening (an indicator of nicotine dependence [USDHHS 1988]), even after the researchers controlled for the number of cigarettes smoked per day (Royce et al. 1993). Conversely, Andreski and Breslau (1993) conducted a study that used the dependence criteria of the *DSM-III*™ and found that, compared with African Americans, greater proportions of whites had symptoms of nicotine dependence. The researchers randomly selected 1,200 adults aged 21–30 years from the members of a health maintenance organization in southeast Michigan. Overall, 22.6 percent of the whites who smoked met the criteria for nicotine dependence, compared with 9.3 percent of the African Americans who smoked. Nicotine dependence was found to have a significant association with psychological distress, as measured by the Brief Symptom Inventory for smokers in both groups. Poor physical health was also associated with nicotine dependence, and this relationship was stronger among African Americans than among whites.

Kandel and colleagues (1997) used questions from the 1991, 1992, and 1993 (combined) National Household Surveys on Drug Abuse (NHSDAs) to develop a proxy measure of *DSM-IV*™ (APA 1994) dependence on various substances (including nicotine). Respondents were asked, for example, if they felt unable to reduce their use when they tried to cut down, experienced withdrawal symptoms (described in this survey as feeling sick because they stopped or cut down), felt that they needed or were dependent on the substance, and felt the need for larger amounts to obtain the same effect. This study used responses from 87,915 persons aged 12 years and older. Among persons who smoked during the previous year, whites were more likely than African Americans, Hispanics, and other racial/ethnic minority group members to be rated as dependent on nicotine. The authors

Table 17. Percentage of adult smokers* who reported that they smoked their first cigarette within 10 minutes and within 30 minutes of awakening, by race/ethnicity and number of cigarettes smoked per day, National Health Interview Survey, United States, 1987

	African Americans		Hispanics		Whites	
Characteristic	%	±CI†	%	±CI	%	±CI
1–14 cigarettes						
≤10 minutes	21.9	4.9	11.3	5.3	11.1	2.1
≤30 minutes	39.2	5.5	26.2	7.3	27.1	3.0
15–24 cigarettes						
≤10 minutes	51.7	8.4	32.7	10.3	36.9	2.4
≤30 minutes	77.6	5.9	61.3	10.3	68.4	2.5
≥25 cigarettes						
≤10 minutes	69.0	18.0	63.3	17.2	61.9	3.0
≤30 minutes	95.6	3.6	93.4	8.2	88.8	1.8

*Persons who reported smoking at least 100 cigarettes in their lives and who reported at the time of survey that they currently smoked.
†95% confidence interval.
Source: National Center for Health Statistics, public use data tapes, 1987.

acknowledged that their study was limited somewhat because the NHSDA indicators of dependence were not based on diagnostic interviews designed specifically to assess *DSM-IV*™ criteria. Nevertheless, the finding that whites were more likely to exhibit indicators of dependence than African Americans was consistent with that of Andreski and Breslau (1993). Further research is needed to resolve the apparent discrepancy for African Americans between studies that are based on the number of minutes to the first cigarette of the day and those that are based on *DSM-III*™ or *DSM-IV*™ criteria for dependence.

Navarro (1996) used population-based data from the 1990 California Tobacco Survey on white (n = 70,997) and Hispanic (n = 28,000) adults. Her analyses indicated that whites were significantly more likely than Hispanics to smoke on a daily basis and to smoke at least 15 cigarettes each day. Furthermore, among the daily smokers, whites were more likely than Hispanics to smoke a cigarette within 30 minutes of awakening. Among Hispanics, those who were less acculturated (i.e., who came from households where the language spoken in the household was not English) were significantly less likely than those who were more acculturated (i.e., who came from households where English was the language spoken) to be daily smokers and to smoke at least 15 cigarettes each day. Among Hispanics who were daily smokers, the percentage who smoked within 30 minutes of awakening did not differ significantly by level of acculturation.

Smoking to maintain a lower body weight is believed to contribute to tobacco dependence. In a survey of high school students in Memphis, Tennessee, Camp and colleagues (1993) found that more whites than African Americans believed that cigarette smoking could help them control their body weight. Among the high school students who smoked, 39 percent of white females and 12 percent of white males reported smoking to control their body weight, compared with none of the African American students.

A few studies have analyzed the perceptions that members of racial/ethnic groups have regarding the addictive nature of tobacco. In a San Francisco area study of 2,835 primary care patients who smoked, Vander Martin and colleagues (1990) found that whites smoked more cigarettes per day and were more likely to consider themselves addicted to cigarettes than African American, Asian American, and Hispanic smokers. Smoking within 15 minutes of awakening was least likely among Hispanic smokers but equally common among smokers in the other groups. In addition, African Americans and Hispanics were less likely than the others to believe that quitting smoking would lead to weight gain.

Most Americans of all races and ethnicities realize that cigarette smoking is addictive. In a survey of 2,092 adults in St. Louis and Kansas City, Missouri, Brownson and colleagues (1992) found that a similar number of whites (90.3 percent) and African Americans (88.5 percent) believed cigarette smoking was addictive. Results from the 1992–1993 CPS (see Chapter 5, Research and Development Limitations) showed that most members of the four racial/ethnic groups as well as whites agreed with the statements that cigarette smoking was an addiction or both a habit and an addiction (Table 18) (U.S. Bureau of the Census, NCI Tobacco Use Supplement, public use data tapes, 1992–1993). Minor differences across gender were observed, although smokers were somewhat less likely to agree with the statements. Approximately 5 percent of the Asian American and Hispanic smokers indicated that cigarette smoking was neither a habit nor an addiction, compared with 1.9 percent of white smokers.

Racial/Ethnic Differences in Quitting Smoking

Because nicotine is addictive, highly addicted smokers have great difficulty in quitting. Differences in quitting can be used as another measure of the level of dependence. Some studies have found that although a similar percentage of whites and African Americans have ever been smokers, the percentage of former smokers has been greater among whites (26.4 percent) than among African Americans (17.2 percent) (Novotny et al. 1988) (see also Chapter 2). Data for 1989 from the BRFSS indicate that the standardized prevalence of smoking cessation was 47 percent among whites vs. 39.1 percent among African Americans (prevalence of cessation was defined as the percentage of ever smokers who were former smokers) (CDC 1990). Similar findings were reported by Kabat and Wynder (1987), Hahn and colleagues (1990), and Geronimus and colleagues (1993). The 1991 NHIS Health Promotion and Disease Prevention supplement collected data on smokers who had quit for at least one day at the time of survey and for at least one month in the previous year (CDC 1993b). Hispanics (52.1 percent) and African Americans (48.7 percent) were more likely than whites (40.3 percent) to have quit smoking for one day. However, data on abstinence from smoking in the previous year showed that Hispanics (16.3 percent) and whites (14.0 percent) were more likely than African Americans (7.9 percent) to have quit smoking for one month or longer. Thus, African Americans were less likely than whites to maintain abstinence. This effect remained after the findings were controlled for socioeconomic status. In an unadjusted analysis of data from the Current Population Survey NCI Supplement, a similar pattern was observed, although the differences between African Americans and whites were slight (see Table 2 and African Americans, Quitting Behavior in Chapter 2).

The lower smoking cessation rates among African Americans do not appear to result from a lack of desire to quit (Royce et al. 1993). In the COMMIT telephone survey, 46.0 percent of African American women and 44.4 percent of African American men stated that they wanted to quit smoking "a lot," compared with 35.0 percent of white women and 33.3 percent of white men. Thus, the lower prevalence of cessation among African Americans may be related to factors other than the desire to quit, such as the absence of culturally appropriate smoking cessation interventions, difficulties in accessing community resources for quitting smoking, and possibly a higher level of nicotine dependence as indicated by comparatively higher levels of cotinine when the data are controlled for the number of cigarettes smoked.

Addiction to Smokeless Tobacco

Considerable nicotine is absorbed from smokeless tobacco. An average systemic dose of nicotine is 3.6 mg for snuff, 4.6 mg for chewing tobacco, and 1.8 mg for cigarettes (Benowitz et al. 1988). Blood nicotine concentrations throughout the day are similar among smokers and those who use smokeless tobacco (Benowitz et al. 1989a). Plasma cotinine levels in regular smokeless tobacco users are often similar to the levels in cigarette smokers (Holm et al. 1992). Abstinence from smokeless tobacco use results in signs and symptoms of nicotine deprivation that are similar to those seen in smokers after they stop smoking (Hatsukami et al. 1987; CDC 1994). These symptoms are reversed by the use of tobacco or administration of nicotine gum. In a study of Swedish oral snuff users, many of the participants considered themselves addicted to snuff, and they reported having as much difficulty giving up smokeless tobacco use as was reported by cigarette smokers trying to quit smoking (Holm et al. 1992). Evidence also suggests that when regular snuff users are deprived of snuff, they will smoke cigarettes to satisfy their need for nicotine (Benowitz 1992b). However, no data are available on racial or ethnic differences in the level of addiction to smokeless tobacco.

Table 18. Percentage of men and women who considered smoking a habit or addiction,* overall and by smoking status, Current Population Survey, United States, 1992–1993

	African Americans		American Indians/ Alaska Natives		Asian Americans/ Pacific Islanders		Hispanics		Whites	
Characteristic	%†	±CI‡	%	±CI	%	±CI	%	±CI	%	±CI
Overall										
Habit	31.7	0.7	19.6	2.6	23.9	1.4	25.1	0.8	17.8	0.2
Addiction	19.8	0.6	19.6	2.6	17.8	1.2	26.3	0.8	21.9	0.2
Both	41.3	0.7	54.6	3.3	46.4	1.6	38.4	0.9	57.0	0.3
Men										
Habit	32.3	1.1	19.5	3.9	25.5	2.0	26.4	1.2	19.3	0.3
Addiction	20.4	0.9	21.4	4.0	18.4	1.8	26.7	1.2	22.0	0.3
Both	39.5	1.1	52.6	4.9	45.8	2.3	36.7	1.3	55.2	0.4
Women										
Habit	31.3	0.9	19.6	3.5	22.5	1.9	24.0	1.0	16.5	0.3
Addiction	19.5	0.8	18.1	3.4	17.2	1.7	25.9	1.1	21.9	0.3
Both	42.5	0.9	56.2	4.4	46.9	2.2	39.8	1.2	58.6	0.4
Nonsmokers										
Habit	29.8	0.8	18.3	3.3	21.7	1.4	23.5	0.8	16.4	0.2
Addiction	20.4	0.7	21.1	3.5	18.9	1.4	27.1	0.9	23.0	0.3
Both	42.9	0.8	54.6	4.2	47.5	1.8	39.4	1.0	57.7	0.3
Men										
Habit	30.3	1.3	19.8	5.3	22.2	2.2	24.6	1.3	18.0	0.4
Addiction	20.5	1.1	22.4	5.5	20.2	2.1	27.9	1.4	22.8	0.4
Both	41.6	1.4	51.4	6.6	48.1	2.6	38.0	1.5	56.1	0.5
Women										
Habit	29.6	1.0	17.3	4.2	21.3	1.9	22.7	1.1	15.0	0.3
Addiction	20.3	0.9	20.2	4.5	17.8	1.8	26.5	1.1	23.1	0.4
Both	43.7	1.1	56.8	5.5	47.0	2.4	40.4	1.3	59.0	0.4
Smokers										
Habit	36.6	1.4	21.5	4.4	36.0	3.9	32.7	2.0	22.1	0.5
Addiction	18.6	1.1	17.5	4.0	12.3	2.7	22.6	1.7	18.9	0.4
Both	37.2	1.4	54.4	5.3	40.9	4.0	34.1	2.0	55.2	0.6
Men										
Habit	36.4	2.0	19.4	5.9	36.6	4.7	32.3	2.5	22.9	0.7
Addiction	20.2	1.7	20.5	6.1	12.6	3.2	23.3	2.3	19.7	0.6
Both	35.1	2.0	53.6	7.5	38.3	4.7	32.8	2.5	53.0	0.8
Women										
Habit	36.7	1.9	23.7	6.4	34.6	7.1	33.2	3.1	21.2	0.7
Addiction	17.2	1.5	14.4	5.3	11.5	4.8	21.4	2.7	18.1	0.6
Both	39.0	1.9	55.2	7.5	47.0	7.5	36.1	3.2	57.3	0.8

*In response to the question, "Do you think smoking is a habit, an addiction, neither, or both?"
†Percentages in this table do not include all categories of responses and thus may not equal 100%.
‡95% confidence interval.
Source: U.S. Bureau of the Census, National Cancer Institute Tobacco Use Supplement, public use data tapes, 1992–1993.

Conclusions

1. Cigarette smoking is a major cause of disease and death in each of the four racial/ethnic groups studied in this report. African Americans currently bear the greatest health burden. Differences in the magnitude of disease risk are directly related to differences in patterns of smoking.
2. Although lung cancer incidence and death rates vary widely among the nation's racial/ethnic groups, lung cancer is the leading cause of cancer death for each of the racial/ethnic groups studied in this report. Before 1990, death rates from malignant neoplasms of the respiratory system increased among African American, Hispanic, and American Indian and Alaska Native men and women. From 1990 through 1995 death rates from respiratory cancers decreased substantially among African American men, leveled off among African American women, decreased slightly among Hispanic men and women, and increased among American Indian and Alaska Native men and women.
3. Rates of tobacco-related cancers (other than lung cancer) vary widely among members of racial/ethnic groups, and they are particularly high among African American men.
4. The effect of cigarette smoking (as reflected by biomarkers of tobacco exposure) on infant birth weight appears to be the same in African American and white women. As reported in previous Surgeon General's reports, cigarette smoking increases the risk of delivering a low-birth-weight infant.
5. No significant racial/ethnic group differences have been consistently demonstrated in the relationship between smoking and infant mortality or sudden infant death syndrome (SIDS); cigarette smoking has been associated with increased risk of SIDS and remains a probable cause of infant mortality.
6. Future research is needed and should focus on how tobacco use affects coronary heart disease, stroke, cancer, chronic obstructive pulmonary disease, and other respiratory diseases among members of racial/ethnic groups. Studies also are needed to determine how the health effects of smokeless tobacco use and exposure to environmental tobacco smoke vary across racial/ethnic minority groups.
7. Persons of all racial/ethnic backgrounds are vulnerable to becoming addicted to nicotine, and no consistent differences exist in the overall severity of addiction or symptoms of addiction across racial/ethnic groups.
8. Levels of serum cotinine (a biomarker of tobacco exposure) are higher in African American smokers than in white smokers for similar levels of daily cigarette consumption. Further research is needed to clarify the relationship between smoking practices and serum cotinine levels in U.S. racial/ethnic groups. Variables such as group-specific patterns of smoking behavior (e.g., number of puffs per cigarette, retention time of tobacco smoke in the lungs), rates of nicotine metabolism, and brand mentholation could be explored.

Appendix. Methodological Issues

It is important to review some methodological issues involved in collecting the data discussed in this chapter. These methodological problems affect the quality of the data and the type of conclusions that can be reached from studies conducted to date. Also, because cigarette smoking tends to be associated with other lifestyle risk factors that impact on health (e.g., Wingard et al. 1982; Vickers et al. 1990; Pérez-Stable et al. 1994), there is a need to control their co-occurrence in order to better understand the health effects of tobacco use.

Classification of Smoking Status

In investigating the health effects of smoking cigarettes and using other tobacco products, researchers typically obtain information from the subjects or surrogate respondents on the use of such products. Questionnaires usually cover cigarette smoking status (i.e., never, former, and current smoker), number of years of smoking and age at initiation of smoking, number of cigarettes smoked per day, and use of other tobacco products (e.g., pipes, cigars, and smokeless

tobacco). However, this information may not be fully valid, resulting in misclassification of exposure to cigarette smoking. A previous report of the Surgeon General reviewed the classification of cigarette smoking status and the consequences of misclassification (USDHHS 1990).

Misclassification of smoking information merits consideration in investigating tobacco use among racial/ethnic populations, because of the potential for bias in comparing the effects of smoking across racial/ethnic groups. To date, such bias has not been identified, although several studies show that Hispanics may underreport cigarette smoking. In a population-based survey in New Mexico, Coultas and colleagues (1988) compared self-reports of smoking against salivary cotinine level (a product of nicotine that has been used as a measure of exposure to nicotine) and end-tidal carbon monoxide concentration. Based on the questionnaire results, the age-standardized prevalence rates of current smoking were 30.9 and 27.1 percent for Hispanic men and women, respectively. After adjusting for cotinine and carbon monoxide levels, these percentages were 39.1 and 33.2. The rate of misclassification was greater in self-reported former smokers than in never smokers, but self-reported never smokers also had levels of cotinine and carbon monoxide indicative of active smoking.

Using information from the Hispanic Health and Nutrition Examination Survey (HHANES), Pérez-Stable and colleagues (1992) documented the misclassification of smoking status through comparisons of self-reports with serum cotinine levels. Among 65 Mexican American former smokers participating in the HHANES in 1982 through 1983, 7 (10.8 percent) had a cotinine level indicative of active smoking; among 124 reported never smokers, 5 (4 percent) were probably active smokers based on their cotinine levels. In a number of surveys, Hispanics, particularly Latino groups in the southwestern and western United States, have been found to smoke about one-half pack of cigarettes per day, compared with non-Hispanic whites who typically report smoking one pack per day (Coultas et al. 1994). Pérez-Stable and colleagues (1992) used data from 547 Mexican American participants in the HHANES to examine underreporting of cigarette consumption using the ratio of serum cotinine to self-reports of the number of cigarettes smoked per day as the "gold standard." This study found that among Mexican Americans, 20.4 percent of men and 24.7 percent of women who were self-reported smokers underreported smoking between one and nine cigarettes per day. Self-reported Mexican American smokers who reported smoking greater numbers of cigarettes per day underreported less frequently.

An analysis of the data from the Coronary Artery Risk Development in (Young) Adults Study (CARDIA) showed that there were higher rates of misclassification in terms of self-reported nonsmokers who had serum cotinine levels of at least 14 ng/mL among African Americans (5.7 percent) than among non-Hispanic whites (2.8 percent) (Wagenknecht et al. 1992). Alternative explanations for underreporting, such as more efficient smoking and differences in cotinine metabolism, could not be excluded.

Two additional studies examined the relationship between ancestry of origin and levels of biochemical markers in smokers. In a study of participants in CARDIA, African American smokers demonstrated higher cotinine levels than non-Hispanic white smokers after controlling for several dimensions of cigarette-smoking behavior (Wagenknecht et al. 1990). Lactose intolerance, which elevates breath hydrogen concentration, may increase the apparent level of expired air carbon monoxide, a readily measured marker of active smoking (McNeill et al. 1990). Lactose intolerance is common in a number of racial/ethnic groups, including Asian Americans and African Americans.

Classification of Race/Ethnicity

The data included in this chapter are derived from diverse sources, including vital statistics, cancer registries, and epidemiological studies on smoking. Race/ethnicity has been classified in these studies using various techniques, including designation on death certificate, classification according to cancer registry protocols, self-reports, birthplace, language use, and surname. The validity of each of these approaches is undoubtedly imperfect; moreover, validity varies across regions and over time. However, comprehensive assessments of the validity of racial/ethnic minority classification in various types of health data have not been reported.

The limited information available indicates some potential for misclassification. For example, Frost and colleagues (1992) compared the classification of "Native American," as recorded by the Seattle-Puget Sound registry of the Surveillance, Epidemiology, and End Results (SEER) Program against an Indian Health Service (IHS) registry of patients eligible for services. A substantial portion of patients with invasive cancer in the IHS registry were not similarly classified by the Seattle-Puget Sound cancer registry. Similarly, an injury registry for the state of Oregon undercounted those with injuries (Sugarman et al. 1993). Using data from the National Longitudinal Mortality Study, Sorlie and colleagues (1992) compared demographic characteristics reported on the CPS of the

U.S. Bureau of the Census with those characteristics reported on the death certificates for persons who died (during a seven-year follow-up period). Among 216 persons identified as American Indians or Alaska Natives by the CPS, only 159 (73.6 percent) were so classified on the death certificate. Similarly, the concordance rate for 272 persons classified by the CPS as Asian Americans or Pacific Islanders was 82.4 percent. Such disagreement suggests that current estimates of mortality rates for selected racial/ethnic groups are underestimated. However, in New Mexico, the classification of "American Indian" by the New Mexico Tumor Registry, also a participant in the SEER Program, closely corresponded with the classification by the state's Bureau of Vital Statistics (Eidson et al. 1994).

Another study in New Mexico also showed a high concordance between self-reported Hispanic race/ethnicity and the designation by the Bureau of Vital Statistics (Samet et al. 1988b). In the report by Sorlie and colleagues (1993), 10.3 percent (n = 62) of persons identified as Hispanics by the CPS were not classified as Hispanics on the death certificate. Surnames also have been used to classify Hispanic ethnicity, using either surname lists developed by the U.S. Bureau of the Census or name recognition algorithms (Howard et al. 1983; Wiggins and Samet 1993). Although studies in parts of the southwestern United States have shown a generally high validity for surname-based approaches for identifying Hispanic ethnicity, the sensitivity and specificity of the various Census Bureau lists have varied over time, and data from the Southwest cannot be readily generalized to other locales. In addition, surname lists tend to exclude women who marry non-Hispanic whites and who take their husband's last name and to exclude as well their children when given the father's non-Hispanic last name (Marín and Marín 1991).

These studies suggest that the validity of classification of race/ethnicity is likely to vary across locations and possibly by type of data. In interpreting health data for racial/ethnic populations, consideration should be given to the potential for misclassification of race/ethnicity and the consequences of any resulting bias.

Classification of Health Outcomes

Comparisons of disease occurrence among racial/ethnic groups also may be biased by differential patterns of disease diagnosis and labeling by race and ethnicity. Such differences may have multiple causes that reflect the complex sequence that begins with the development of symptoms and signs and extends to the labeling of an illness by a clinician or the statement of cause-of-death on a death certificate. Health beliefs and knowledge, ability to access and pay for medical care, the quality of care available, and differential patterns of care by race/ethnicity may all affect diagnoses of illnesses. A full review of these topics is beyond the scope of this report, but several examples are offered to illustrate the potential for differential patterns of classification of health outcomes by race/ethnicity.

Becker and colleagues (1990) examined the assignment of underlying cause of death to the category "symptoms, signs, and ill-defined conditions" in the *Manual of the International Classification of Diseases, Injuries and Causes of Death* (*ICD*). In the nation, the crude death rate for this non-specific category has paralleled the mortality rate in this category for African Americans. Becker and colleagues (1990) analyzed vital statistics data for New Mexico for 1958 through 1982 and calculated mortality rates for "symptoms, signs, and ill-defined conditions" by racial/ethnic group. The state mortality rates for Hispanics, non-Hispanic whites, and American Indians for this category exceeded the nationwide rates. Among the racial/ethnic minority groups in New Mexico, American Indians had particularly high mortality rates; for men, 8.4 percent of American Indian deaths were in this category versus 5.9 percent of Hispanic deaths and 5.0 percent of non-Hispanic white deaths. Similarly, mortality rates for cancers of ill-defined and unknown primary sites tend to be much higher in American Indians in several areas of the country than for all racial/ethnic groups combined (Valway 1992).

Recent comparisons of the evaluation and management of chest pain and coronary artery disease in African Americans and non-Hispanic whites further illustrate the potential for bias by race/ethnicity in diagnostic classification. In a study of patients presenting to an emergency room with chest pain, African Americans were less likely to be admitted and less likely to be sent to a coronary care unit once they were admitted (Johnson et al. 1993). The study also found that African Americans were as likely as non-Hispanic whites to have cardiac catheterization. In contrast, other studies, using Department of Veterans' Affairs, Medicare, and other large data bases, have shown that African Americans are less likely than non-Hispanic whites to have cardiac catheterization and invasive interventions for coronary artery disease (Wenneker and Epstein 1989; Udvarhelyi et al. 1992; Ayanian et al. 1993; Franks et al. 1993; Whittle et al. 1993; Peterson et al. 1994). These differential patterns of evaluation by race/ethnicity could introduce bias in investigations of tobacco smoking and coronary artery disease among African Americans and non-Hispanic whites by underestimating the effects of cigarette smoking on coronary artery disease.

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